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RESEARCH ARTICLE OPEN ACCESS

Prevalence, Risk Factors and Transmission of Carbapenemase-producing Organisms in Mothers and Neonates in Women Hospital

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Abstract

A study in the Neonatal Department of Maternity Hospital in HCM, Vietnam, in 2019 reported that the prevalence of Carbapenemase-producing *Enterobacteriaceae* (CPE) was 42.20% (n = 83). However, risk factors of CPE colonization and transmission were still an unsolved question. Hence, we implemented this study. A prospective study was conducted from April to July 2020 at the Childbirth Ward of Hung Vuong Hospital, where 359 pairs of mothers and their neonates participated in our research. We applied laboratory methods to confirm CPE colonization and its antibiotic resistance, including rectal swab tests, chromo-carba plates, MALDI-TOF method, antibiograms, and rep-PCR method. The 23.0 version of SPSS was a software to analyze personal characteristics, prevalence, and risk factors for CPE colonization. Adjusted odds ratio and 95% confidence interval were considered significant at P < 0.05. The results showed that the prevalence of CP *E. coli* transmission between mothers and neonates was 0.28% (1/359), confirmed by the rep-PCR method. The characteristics that reduced the CPE-colonization risks in mothers were the mother's age (19-23 years old), vaginal delivery, mothers caring for neonates, skin-to-skin contact time, and breastfeeding. However, the risk factors that increased the CPE colonization in neonates were the NICU admission before fecal sampling and the number of vaginal examinations performed on mothers before delivery. Although the prevalence of mother-to-neonate CPE transmission was low, screening for CPE colonization at hospital admission, adhering to hand hygiene, and implementing aseptic medical practices are crucial standards for preventing and controlling CPE colonization in the healthcare sector.

Keywords: CPE, Neonates, Mothers, Risk Factors, Mother-to-Neonate CPE Transmission

INTRODUCTION

The third-generation cephalosporins, in the early 1980s, were the best choice against lactamase-producing bacteria. However, their effectiveness was reduced by plasmid-encoded lactamases, as shown in the first report in 1983.[1] From 1989 to 2004, extended-spectrum β-lactamases (ESBLs) spread in Europe, North America, South and Central America, Africa, the Middle East, Australia, and Asia.[2] In early 1990, IMP carbapenemases, called the first plasmid-mediated transferable carbapenemases, emerged in Japan.[3] Continuously, carbapenem-resistant *Enterobacteriaceae* (CRE) or carbapenemase-producing *Enterobacteriaceae* (CPE) existed in many countries worldwide, including high-income countries,[4,5] and, secondly, in lower and middle-income countries.[6] All spreading drug-resistant clones detected in carriers are in the commensal microflora.[7] CRE colonization is most often in the gastrointestinal tract of carriers.[8] A recent systematic review reported 1,806 patients colonized with CRE at the time of admission. It implied that CRE existed in the community, and this study also found that 299 (16.5%) acquired the clinical infection during hospital stay.[9]

In 49 Asian countries that participated in the CRE study, three countries reported the highest resistance rates to imipenem: Indonesia (5.8%), Vietnam (3.0%), and the Philippines (3.7%).[9-13] A study of "Prevalence and risk factors of carbapenemase-producing organisms in a Neonatal Department of Maternity Hospital in the South of Vietnam" from January to March 2019, with 83 neonates chosen randomly, showed the prevalence of CPO was 42.20% (P470).[14] The questions are whether CPO may be transmitted from mothers to neonates in Hung Vuong hospital, and what are the risk factors of CPO colonization in mothers and their neonates? It is the reason we conducted this study.

MATERIALS AND METHODS

Method

Definition of carbapenemase-producing bacteria

Carbapenemase-producing *Enterobacteriaceae* (CPE) are microorganisms belonging to the group of *Enterobacteriaceae* that are unsusceptible to carbapenems through producing a carbapenemase enzyme.[12] It is usually, but not always, through the production of carbapenemase.

CRE are *Enterobacteriaceae* resistant to any carbapenem antimicrobial whose minimum inhibitory concentrations are ≥4 mcg/ml for doripenem, meropenem, or imipenem, or ≥2 mcg/ml for ertapenem; or documented to produce carbapenemase (CPE).[8] Clinically, CRE are screened with MELAB Chromogenic CARBA and then confirmed with the rapid BD Phoenix system.

Design and data collection

Study design

This prospective cohort study was conducted from April to July 2020 at Hung Vuong Hospital, specifically in the Delivery Department, where we collected fecal samples from mothers approximately 24 hours after admission to the hospital. Additionally, we also collected neonate fecal samples in the Post-Delivery or Neonatal Department of neonates. The length of time for the fecal sample collection in neonates was about 24 to 48 hours after delivery. There are approximately 3,600 pregnant women admitted to the Delivery Department every month. It means the number of neonates born here was approximately 3600 for one month. Mothers admitted into the Delivery Department were chosen randomly in our study. The children of these mothers were subjects invited to participate in our research. The sample size of mothers can be calculated with the Yamane formula[15] as follows:

$$n = \frac{3,600}{1+Ne^2} = \frac{3,600}{1+3,600.(0.05)^2} = 360$$ [15]

and the sample size for neonates was also 360

Where: n = the sample size, N = the population size, e = the acceptable sampling error (95% confidence level and P = 0.05 are assumed).

Conceptual Framework and Flow Diagram of the study is shown in Figure 1.

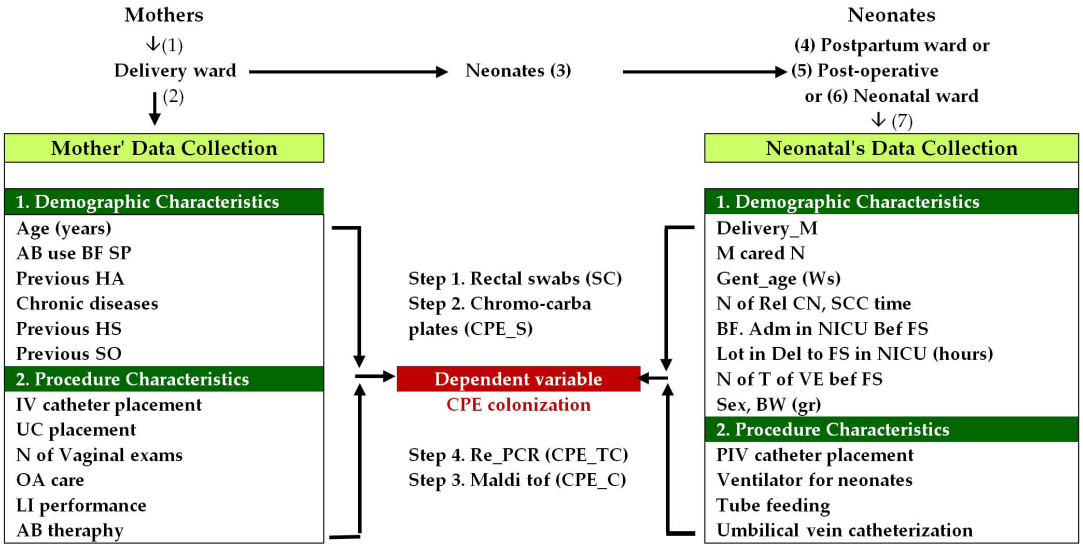


Figure 1. Conceptual Framework and Process of Conducting the study of Prevalence and risk factors of CPE in mothers and neonates and transmission of carbapenemase producing organisms in Women Hospital
ABU: Antibiotic use, AB: Antibiotic, Adm in NICU Bef FS: Admission in NICU before fecal sampling, BF: Breast feeding, BW (gr): Birth Weight (gr), CPEC: CPE confirmation, CPES: CPE screening, CPE_TC: CPE transmission, confirmation, Del M: Delivery method, GA (Ws): Gestational age (Ws), HA: Hospital admission, HS: Hospital stay, LI: Labor induction, LoT fr Del to FS in NICU (hrs.): Length of time from delivery to fecal sampling in NICU (hours), MCN: Mother cared Neonates, N: Number, N of Rel CN: Number of Relatives cared Neonates, N of T of VE Bef FS: Number of times of Vaginal examination before fecal sampling, OA: Obstetric Analgesia, PIV: Peripheral intravenous catheter, SC: Sample collection, Sex, SO: Surgical operation, SP: Study participation, SSC time: Skin to skin contact time. UC: Urinary catheters

Inclusion criteria

All mothers who were admitted and delivered their babies at the Delivery department in Hung Vuong Hospital from April to July 2020 were subjects in this study.

Exclusion criteria

If mothers did not wish to participate in this study, they and their babies would not be the subjects in this study, and neonates had an abnormal anus or did not have an anus.

One neonate did not continue participating in our study because this neonate was transferred to the Children's Hospital on the first day after birth to be treated by a neonatal specialist. Hence, the number of mothers and neonates participating in our study was 359 for each group.

Material

Data collection

We collected the study data from the medical records,

a. Independent variables collected for mothers included characteristics: (a) demographics such as age (years), prior antibiotic treatment, prior hospital admission, chronic disease, prior hospital stay, prior surgical intervention[1,16]; and (b) procedures performed on mothers,[16,17] such as peripheral intravenous catheter and urinary catheter use;
b. Independent variables collected for neonates included characteristics: (a) demographics[1,16] such as sex, gestational age (weeks), age (days), birth weight (kgs), length of stay (days), prior exposure to antibiotics, Cesarean section/Vaginal delivery; and procedure characteristics performed on neonates,[1,16] such as invasive device: peripheral intravenous catheter, Mechanical ventilation,[2,16] feeding tube and umbilical vein catheterization use.
c. The dependent variable was CPE for mothers and neonates.

Conceptual Framework and Flow Diagram of Study

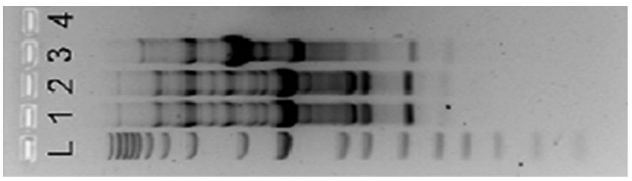
Microbiology methods

Tools to screen and detect CPE include: First, we utilized the rectal swabs (Nantong Renon Laboratory Equipment Co., Ltd., No. 128, Xiaohai Road, Sanhe Town, Haimen, Jiangsu) to collect the fecal samples. Then, we transported these samples to the Lab Unit of OUCRU, Oxford University Clinical Research Unit, in HCM City, to screen CPE by ChromID carba plates produced with the selective medium called Chromagar (29 Av. George Sand, 93210 Saint-Denis, France). Next, we used the MALDI TOF method (Bruker Daltonics GmbH & Co. KG, Fahrenheitstraße 428359 Bremen, Germany) for species identification of colonies on blood agar. At the end of step one, we use antimicrobial susceptibility testing to confirm the antibiotic resistance of the species.

In step two, we determine the association between mothers and their children by first displaying the genome patterns of mothers and neonates with the rep-PCR DNA fingerprinting technique (CD Genomics, SUITE 111, 17 Ramsey Road, Shirley, NY 11967, USA). Secondly, we perform whole-genome sequencing to investigate this association, if possible.

Statistical analysis

23.0 version of SPSS software was a tool used to analyze data. Descriptive statistics analyzes characteristics associated with demographics, procedures performed on mothers and neonates, and the prevalence of CPE. Multivariate logistic regression analyzes the risk factors related to CPE and non-CPE status, and the adjusted odds ratio with a 95% confidence interval was considered significant. The level of statistical significance was $P < 0.05$.



Rep-PCR review

Lane 4: Negative control
Lane 3: E. coli 25922
Lane 2: CP E. coli from Mother sample (116B)
Lane 1: CP E. coli from Neonate sample (116B)
L: DNA Ladder 1 kb plus, Invitrogen

Figure 2. Comparison of CPE of mother and neonates based on result of rep-PCR

Table 1. Prevalence of CPE in mothers and neonates

CPE 1 in Mother	Freq (%)	CPE 2 in Mother	Freq. (%)	% Mother CPE	CPE in Neonates	Freq. (%)	% Neonate CPE	% CP. *E. coli* transmission between M&C
E. coli	16 (4.45)			5.85 (21/359)	*E. coli*	5 (1.39)	3.62 (13/359)	0.28 (1/359)
	3 (0.84)	*K. pneumoniae*	1 (0.28)		*K. pneumoniae*	5 (1.39)		
		K. pneumoniae	1 (0.28)		*Ent. cloacae*	2 (0.56)		
		Ent. cloacae	1 (0.28)		*Ent. kobei*	1 (0.28)		
K. pneumoniae	2 (0.56)	*E. coli*	1 (0.28)					
Non-CPE	338 (94.15)	Non-CPE	355 (98.88)		Non-CPE	346 (96.38)		
Total	359 (100)		359 (100)		Total	359 (100)		

RESULTS

Prevalence of CPE colonization in mothers and neonates

The study result showed twenty-one mothers colonized by CPE, including nineteen *E. coli* and two K. pneumoniae, in whom four mothers colonized by two CPE included three mothers colonized by *E. coli* and (*K. pneumoniae* or *Ent. cloacae*), and one colonized by *K. pneumoniae* and *E. coli*. Hence, the prevalence of CPE colonization in mothers and neonates was 5.85% (21/359), and 3.62% (13/359, respectively, as shown in Table 1, while carbapenem-producing *K. pneumoniae* (KPC) appeared in neonates.

CPE transmission between mothers and neonates

Our study findings showed that the CPE of a child was the same as that of a mother, a 32-year-old teacher, who was admitted to the Maternity Hospital approximately 86 hours before study participation, and the Delivery department for 6 hours before collecting feces by rectal swab. This 32-year-old mother had no risk of CPE colonization before our study participation, including the length of stay in hospital, the past year, antibiotic use, and nosocomial infections due to CPE or non-CPE before participating in this study. This mother gave birth vaginally and breastfed her 37 week-gestational age and 2350 gram-weight male baby, who spent skin-to-skin contact with her mother for two hours, did not take antibiotics before taking fecal sampling, was not admitted to the Neonatal Department, and was taken care of by his mother and father.

Based on the method of rep-PCR, we confirmed that the CPE of the mother was identical to that of her neonate, as shown in Figure 2. It means that the mother transmitted CP. *E. coli* to her child. Hence, the prevalence of CP *E. coli* transmission between a mother and her child was 0.28 (1/359) (Table 1).

Risk factors of CPE colonization in mothers

Univariate binary logistic regression of risk factors of CPE colonization in mothers

Our study research also showed that the total doses of antibiotics consumed by mothers before fecal sample collection were risk factors for CPE colonization, compared with mothers

Table 2. Univariate Binary Logistic Regression of Risk Factors of CPE Colonization in mothers in the Delivery Department of Maternity Hospital, April to July 2020

Cell		Code	B	S.E.	Wald	df	Sig.	Exp(B)	95% CI for EXP(B)	
									Lower	Upper
1	ABU before study participation	Non-ABU: (0), ABU: (1)	-0.115	0.083	1.936	1	0.164	0.891	0.757	1.048
		Constant	-1.831	0.695	6.937	1	0.008	0.160		
2	Total AB doses used by Mother before fecal sample collection	T of ABD = 0 dose			10.961	2	0.004			
		T of ABD ≤2 doses (1)	1.949	0.858	5.158	1	0.023	7.022	1.306	37.755
		T of ABD ≥3 doses (2)	1.661	0.618	7.220	1	0.007	5.267	1.568	17.695
		Constant	-3.048	0.264	133.013	1	0.000	0.047		
3	ADM before study participation about one year	Non-ADM: (0), ADM:(1)	0.396	1.070	0.137	1	0.711	1.486	0.183	12.093
		Constant	-2.794	0.230	147.154	1	0.000	0.061	2	
4	Mother's age (years)	≤18			4.955	3	0.175			
		19-23	-2.702	1.266	4.554	1	0.033	0.067	0.006	0.802
		24-28	-1.030	0.835	1.520	1	0.218	0.357	0.070	1.835
		≥29	-0.727	0.853	0.726	1	0.394	0.484	0.091	2.571
		Constant	-1.705	0.769	4.918	1	0.027	0.182		
5	Number of days for hospital stay in the last ADM	Non-Hospital stay (0)			0.844	2	0.656			
		<2 weeks (1)	1.015	1.104	0.844	1	0.358	2.758	0.317	24.028
		1-2 months	-18.397	40192.970	0.000	1	1.000	0.000	0.000	
		Constant	-2.806	0.230	148.541	1	0.000	0.060		
6	Duration of Hospital stay before sample collection (hours)	≤24			6.390	2	0.041			
		24-48	1.036	0.602	2.961	1	0.085	2.819	0.866	9.177
		≥48	1.335	0.611	4.765	1	0.029	3.799	1.146	12.595
		Constant	-3.084	0.284	118.234	1	0.000	0.046		
7	Chronic diseases	Non-Chronic diseases (0), Chronic diseases (1)	-18.504	7882.490	0.000	1	0.998	0.000	0.000	
		Constant	-2.698	0.225	143.274	1	0.000	0.067		
8	Past surgical operation Before study participation	Non-PSO (0) PSO (1)	-0.511	1.045	0.239	1	0.625	0.600	0.077	4.651
		Constant	-2.747	0.231	141.857	1	0.000	0.064		
9	Professional	HCW (0)			0.311	4	0.989			
		State employee (1)	18.513	14210.307	0.000	1	0.999	109692359.845	0.000	
		Businessperson (2)	18.430	14210.307	0.000	1	0.999	100966831.221	0.000	
		Self-employed (3)	18.677	14210.307	0.000	1	0.999	129237543.962	0.000	
		Stay-at-home spouse (4)	18.258	14210.307	0.000	1	0.999	85024699.975	0.000	
		Constant	-21.203	14210.307	0.000	1	0.999	0.000		

Table 2. Cont...

Cell		Code	B	S.E.	Wald	df	Sig.	Exp(B)	95% CI for EXP(B)	
									Lower	Upper
10	Duration of performance of profession (years)	Duration of work	0.069	0.048	2.040	1	0.153	1.071	0.975	1.177
		Constant	-3.074	0.326	88.970	1	0.000	0.046		
11	Obstetric Analgesia	OA: Y (1), N (0)	0.111	0.464	0.058	1	0.810	1.118	0.451	2.773
		Constant	-2.820	0.285	97.532	1	0.000	0.060		
12	Length of time from Obstetric Analgesia to Sample collection	Obstetric Analgesia (1), Non-Obstetric Analgesia (0)	0.028	0.109	0.066	1	0.797	1.029	0.830	1.275
		Constant	-2.803	0.247	128.934	1	0.000	0.061		
13	Labor induction	Yes (1), No (0)	2.064	0.731	7.970	1	0.005	7.881	1.880	33.040
		Constant	-2.912	0.242	144.738	1	0.000	0.054		
14	Length of time from Labor induction to Sample collection (hours)	LoT fr LI to SC ≤24 hrs.			10.740	3	0.013			
		LoT_fr_LI_to_SC from > 24-36 hrs. (1)	-18.288	28420.722	0.000	1	0.999	0.000	0.000	
		LoT fr LI to Sc from 36-48 hrs. (2)	2.915	1.435	4.127	1	0.042	18.444	1.108	307.005
		LoT fr LI to SC >48 hrs. (3)	2.509	0.944	7.060	1	0.008	12.296	1.932	78.280
		Constant	-2.915	0.242	145.060	1	0.000	0.054		

Table 3. Multivariate Binary Logistic Regression of Risk Factors of CPE Colonization in mothers in the Delivery Department of Maternity Hospital, April to July 2020

Characteristics	Code	B	S.E.	Wald	df	Sig.	Exp(B)	95% CI for EXP(B)	
								Lower	Upper
Age (years)	≤18.00			3.761	3	0.288			
	19.00-23.00	-2.159	1.353	2.548	1	0.110	0.115	0.008	1.635
	24.00-28.00	-0.572	0.983	0.339	1	0.561	0.564	0.082	3.877
	≥29.00	-0.164	1.005	0.027	1	0.870	0.849	0.118	6.086
Delivery method (C)	Vaginal delivery (1), Cesarian section (0)	1.398	0.767	3.317	1	0.069	4.045	0.899	18.201
Mother care Child (C)	M cared C (1), M_n0 Cared C (0)	-1.031	0.777	1.758	1	0.185	0.357	0.078	1.637
Length of time of	t ≤ 24.00			2.880	2	0.237			
Hospital stay before	24 < t ≤ 48	1.111	0.656	2.869	1	0.090	3.038	0.840	10.993
sample collection	t > 48	0.152	1.117	0.018	1	0.892	1.164	0.130	10.394
(hours)									
Length of time from	LoT fr LI to SC: t ≤24			2.922	3	0.404			
Labor induction to	LoT fr LI to SC: 24 < t ≤ 36	-18.390	27704.599	0.000	1	0.999	0.000	0.000	
sample collection	LoT fr LI to SC: 36 < t ≤ 48	3.469	2.038	2.897	1	0.089	32.119	0.591	1744.745
(hours)	LoT fr LI to SC: t > 48	1.136	1.597	0.506	1	0.477	3.115	0.136	71.308
Total of Antibiotic	Zero dose			5.000	2	0.082			
doses consumed by	1-2 doses	1.674	0.749	5.000	1	0.025	5.336	1.230	23.153
mother before sample	≥3 doses	0.186	1.560	0.014	1	0.905	1.205	0.057	25.634
collection									
	Constant	-2.830	1.230	5.296	1	0.021	0.059		

Table 4. Univariate Binary Logistic Regression of Risk Factors of CPE Colonization in Neonates at the Post Delivery Department of Maternity Hospital, April to July 2020, April to July 2020

Cell	Characteristics	Code	B	S.E.	Wald	df	Sig.	Exp(B)	95 % CI for EXP(B)	
									Lower	Upper
1	Delivery method	Caesarean section: (0), Vaginal delivery: (1),	-1.427	0.583	6.000	1	0.014	0.240	0.077	0.752
		Constant	-2.485	0.368	45.598	1	0.000	0.083		
2	Mother cared Child	Mother no cared child: (0), Mother cared Child: (1)	-1.909	0.582	10.770	1	0.001	0.148	0.047	0.464
		Constant	-1.872	0.439	18.219	1	0.000	0.154		
2*	Delivery method by mother cared Child	Delivery method by mother cared Child	-2.886	1.258	5.260	1	0.022	0.056	0.005	0.657
		Constant	-2.313	0.332	48.660	1	0.000	0.099		
3	Gestational age (Weeks)	Gest age <37Ws: (0) Gest age ≥37 Ws: (1),	-1.175	0.588	3.984	1	0.046	0.309	0.097	0.979
		Constant	-2.416	0.467	26.791	1	0.000	0.089		
4	Number of relative cared child	N of Rel car Child: (0)			16.311	2	0.000			
		N of Rel car Child ≤2 (1)	-3.908	0.968	16.311	1	0.000	0.020	0.003	0.134
		N of Rel car Child ≥3 (2)	-21.608	11602.711	0.000	1	0.999	0.000	0.000	
		Constant	0.405	0.913	0.197	1	0.657	1.500		
5	Skin to skin contact time (hours)	SSC time = (0)			9.830	2	0.007			
		SSC time = (1)	-0.847	1.101	0.592	1	0.442	0.429	0.049	3.711
		SSC time = (2)	-1.892	0.603	9.830	1	0.002	0.151	0.046	0.492
		Constant	-2.097	0.401	27.419	1	0.000	0.123		
6	Breast feeding	Breast Feed Yes: (1), Breast Feed No: (0)	-1.699	0.635	7.171	1	0.007	0.183	0.053	0.634
		Constant	-1.872	0.537	12.146	1	0.000	0.154		
7	Admission in NICU before fecal sampling	Adm in NICU B Fecal sample: (1), No Adm in NICU B fecal sample: (0)	3.069	0.712	18.582	1	0.000	21.524	5.332	86.888
		Constant	-3.629	0.338	115.447	1	0.000	0.027		
8	Length of time from delivery to fecal sampling in NICU (hours)	L of T fr del to Fecal sample in NICU: 0 hr. (1),			18.599	2	0.000			
		L of T fr del to Fecal sample in NICU 17.00 to 36.00 hrs (2)	3.223	0.973	10.967	1	0.001	25.111	3.727	169.190
		L of T fr del to Fecal sample in NICU: >36 hrs (3)	2.936	0.930	9.974	1	0.002	18.833	3.046	116.456
		Constant	-3.629	0.338	115.447	1	0.000	0.027		

Table 4. Cont...

Cell	Characteristics	Code	B	S.E.	Wald	df	Sig.	Exp(B)	95 % CI for EXP(B)	
									Lower	Upper
9	Number of times of Vaginal examination before fecal sampling	No of Vag. exam: from 1 to 5 times: (0)			8.311	2	0.016			
		No of Vag. exam: from 6 to 9 times: (1)	2.531	1.052	5.795	1	0.016	12.571	1.601	98.737
		No of Vag. exam: from ≥ 10 times: (2)	0.639	1.421	0.202	1	0.653	1.895	0.117	30.716
		Constant	-4.970	1.003	24.529	1	0.000	0.007		
10	Sex	Male: (1), female: (2),	0.258	0.567	0.208	1	0.649	1.295	0.426	3.931
		Constant	-3.412	0.415	67.631	1	0.000	0.033		
11	Weight (grams)	≤2500 grs: (1), >2500 grs: (2)	-1.322	0.810	2.661	1	0.103	0.267	0.054	1.305
		Constant	-0.758	1.531	0.245	1	0.621	0.469		
12	Delivery method	Caesarean section: (0), Vaginal delivery: (1),	1.224	0.990	1.529	1	0.216	3.400	0.489	23.652
	Mother cared Child	Mother no cared child: (0), Mother cared Child: (1)	-0.601	0.739	0.662	1	0.416	0.548	0.129	2.332
	Delivery method by Mother care Child	Delivery method by Mother care Child	-2.886	1.258	5.260	1	0.022	0.056	0.005	0.657

Table 5. Multivariate Binary Logistic Regression of Risk Factors of CPE Colonization in neonates in the Delivery Department of Maternity Hospital, April to July 2020

Characteristics	Codes	B	S.E.	Wald	df	Sig.	Exp(B)	95% C.I. for EXP (B)	
								Lower	Upper
1. Delivery method	Caesarean section: (0), Vaginal delivery: (1),	-20.583	15563.029	0.000	1	0.999	0.000	0.000	
2. Mother cared child	Mother no cared child: (0), Mother cared Child: (1)	0.176	0.997	0.031	1	0.860	1.193	0.169	8.412
3. Delivery method by Mother cared Child	Delivery method by Mother care Child	20.362	15563.029	0.000	1	0.999	696910049.542	0.000	
4. Number of Relative cared child	N of Rel cared Child: (0), N of Rel cared Child: ≤2 (1), N of Rel cared Child: ≥3 (2)	-22.926	15563.029	0.000	1	0.999	0.000	0.000	
5. Gestational age (Weeks)	Gest age <37Ws: (1) Gest age ≥37 Ws: (2),	-0.692	0.955	0.525	1	0.469	0.501	0.077	3.254
6. Skin to skin contact time (hours)	SSC time = (0)			1.763	2	0.414			
	SSC time = (1) (1)	-0.575	1.353	0.181	1	0.671	0.562	0.040	7.978
	SSC time = (2) (2)	-1.988	1.498	1.763	1	0.184	0.137	0.007	2.578
7. Breastfeeding	Breast Feed Yes: (1), Breast Feed No: (0)	-0.016	1.142	0.000	1	0.988	0.984	0.105	9.223
8. Length of time from delivery to fecal sampling in NICU (hours)	LoT from NICU ADM to SC: 0 (hour)			0.865	2	0.649			
	LoT from NICU ADM to SC ≤36 (1)	2.667	3.131	0.726	1	0.394	14.392	0.031	6649.807
	LoT_from NICU ADM to SC: >36 (2)	1.747	2.144	0.664	1	0.415	5.738	0.086	383.637
9. Number of times of Vaginal examination before fecal sampling	No of Vag exam from 1-5 times: (0)			5.466	2	0.065			
	No of Vag exam: from 6-10 times: (1)	2.673	1.308	4.174	1	0.041	14.487	1.115	188.237
	No of Vag exam: from ≥11 times: (2)	1.128	1.620	0.484	1	0.486	3.088	0.129	73.966
10. Neonatal Birth weight (gram)	Neonatal Birth weight: ≤2500 grs (0), Neonatal Birth weight >2500 grs (1)	-1.072	1.342	0.638	1	0.424	0.342	0.025	4.748
11. Sex	Male (1), Female (0)	0.384	0.721	0.284	1	0.594	1.469	0.358	6.035
	Constant	20.590	15563.029	0.000	1	.999	874888019.484		

who did not consume antibiotics. For example, the groups of mothers with total antibiotic doses less than two or more than three doses had the risk of CPE colonization more than 7 or 5 times compared with the group of mothers not using antibiotics, with P = 0.023, OR = 7.022, 95% CI: 1.306-37.755, and P = 0.007, OR = 5.267, 95% CI: 1.568-17.695, respectively. The length of time from labor induction to sample collection, from 36 to 48 hours, and over 48 hours, was a risk factor associated with CPE colonization more than 18 and 12 times, compared with the group of mothers with the length of time from labor induction to sample collection less than 24 hours, as described in Table 2, with P = 0.042, OR = 18.444, 95% CI: 1.108-307.005 and P = 0.008, OR = 12.296, 95% CI: 1.932-78.280, respectively. The length of time from labor induction to sample collection was over 48 hours, which meant mothers spent more than 48 hours in the hospital.

Multivariate binary logistic regression of risk factors of CPE colonization in mothers

When analyzing the multivariate binary logistic regression of risk factors associated with CPE colonization in mothers, we determined that the total antibiotic dose from 1 to 2 doses, consumed by mothers before sample collection was a principal risk factor of CPE colonization, and the group of mothers consuming the antibiotic doses from 1 to 2 doses before sample collection, was colonized by CPE colonization with more than 5.3 times, with P = 0.025, OR = 5.336, 95% CI: 1.230-23.153, compared with the rest group of mothers (Table 3).

Univariate binary logistic regression of risk factors of CPE colonization in neonates

By the univariate binary logistic regression of risk factors of CPE colonization in the neonates at the Post Delivery Department of Maternity, we detected that the vaginal delivery, mother caring for her neonate, and the interaction of these two factors, one positive factor, reduced the risks of CPE colonization in neonates, with P = 0.014, OR = 0.240, 95% CI: 0.077-0.752, in the Cell 1; P = 0.001, OR = 0.148, 95% CI: 0.047-0.464, in the Cell 2; and P = 0.022, OR = 0.056, 95% CI: 0.005-0.657, in the Cell 2* of the Table 4, respectively. The other beneficial factors reducing the CPE colonization in neonates included neonates' gestational age (weeks), with P = 0.046, OR = 0.309, 95% CI: 0.097 -0.979, in The Cell 3 of Table 4, and especially, the number of relatives cared neonates less than two relatives, with P = 0.000, OR = 0.020, 95% CI: 0.003-0.134, in the Cell 4 of Table 4. One advantageous factor associated with decreasing the risk of CPE colonization was the skin-to-skin contact time between mothers and neonates for two hours, with P = 0.002, OR = 0.151, 95% CI: 0.046-0.492, in the Cell 5 of Table 4 and mothers breastfeeding their neonates prevented and reduced CPE colonization in her neonates, with P = 0.007, OR = 0.183, 95% CI: 0.053-0.634, In the Cell 6 of Table 4.

Along with positive factors in our study, we also found negative features associated with CPE colonization in neonates. For instance, neonates admitted to the NICU before fecal sampling had a risk of CPE colonization compared with neonates not admitted to the NICU, with P = 0.000, OR = 21.524, 95% CI: 5.332-86.888, as shown in Cell 7 of Table 4.

In addition, we recognized the lengths of time from delivery to fecal sampling in NICU from 17-36.00 hours, and over 36.00 hours were the risk factors of CPE colonization, compared with the group of neonates collected with fecal samples at the beginning of NICU admission, with P = 0.001, OR = 25.111, 95% CI: 3.727-169.190, and P = 0.002, OR = 18.833, 95% CI: 3.046-116.456, respectively, as shown in Cell 8 of Table 4.

Finally, vaginal examination before fecal sampling was a risk factor for CPE colonization when the number of vaginal examinations was from 6 to 10 times compared to that from 1 to 5 times, with P = 0.016, OR = 12.571, 95% CI: 1.601 -98.737, as shown in Cell 9 of Table 4.

Multivariate binary logistic regression of risk factors of CPE colonization in neonates

The result of multivariate binary logistic regression of risk factors of CPE colonization in neonates showed the number of vaginal examination times before fecal sampling from 6-10 times was the principal risk factor of CPE colonization, which influenced all other risk factors detected in mothers that had vaginal birth,

Table 6. Characteristics associated with CPE in mothers and neonates

OCCUP (1)	Study Participants (2)	CPE_1 (3)	CPE_2 (4)	M & C had CPE ID (5)	CPE_ HI (6)	Non-CPE HI (7) (Case)	Non-HI (8)	ABU Bef Part Study (9) (Case)
HCWs (n = 8)	M	0	0	No	0	0	0	0
	Nn	0	0		0	0	0	
State Employee (n = 173)	M	*E. coli:* ten *K. pneumoniae:* one	*E. coli:* one *Ent. cloacae:* one	Yes*: E. coli*	0	SSI = 1 PWI = 1	0	11
	Nn	*E. coli:* one *K. pneumoniae:* two *Ent. cloacae:* one *Ent. kobei:* one			0	0	NI	
Businessperson (n = 51)	M	*E. coli:* two *K. pneumoniae:* one	*K. pneumoniae:* one	No	0	0	0	4
	Nn	*E. coli:* two *Ent. cloacae:* one			0	0	0	
Self-employed (n = 27)	M	*E. coli:* two		No	0	0	0	2
	Nn	*E. coli:* one			0	0	0	
Stay-at-home spouse (n = 100)	M	*E. coli:* five	*K. pneumoniae:* one	No	0	0	0	7
	Nn	*E. coli:* one *K. pneumoniae:* three			0	0	0	

C: Child, CPE ID: CPE Infectious disease, HI: Hospital infection, M: Mother, NI: Neonatal infection, Nn: Neonate, OCCUP: Occupation, PWI: Perineal wound infection, SSI: Surgical site infection

Table 6. Characteristics associated with CPE in mothers and neonates (Cont.1)

Occup. (1)	Study Participants (2)	Last ABU Bef Adm, HL Ago fr Adm (10) (Month)	M cared C (11) (%)	Rel cared C (12) (%)	GA (13) (Weeks)	NICU adm Bef SC (14)	Reas for NICU ADM (15) (Case)	Deliv M - VAG Del (16) (Case) (%)
HCWs (n = 8)	M		87.50%					
	Nn			100%	36-40	0	0	75%
State Employee (n = 173)	M	≤3 mos (4/173) ≥6 mos (6/173)	86.71%					
	Nn			98.84%	35-41	4	NeoJ: seven, RF: two PTB: one NI: two	72.30%
Businessperson (n = 51)	M	≥9 mos (4/51)	86.30%					
	Nn			96.10%	33-40	3	NeoJ: two, RF: two PTB: one, BLDS: one,	60.80%
Self-employee (n = 27)	M	≤3 mos (1/27) ≥9 mos (1/27)	81.50%					
	Nn			100%	32-41	1	PTB: one,	66.66%
Stay-at-home spouse (n = 100)	M	≤3 mos (1/100) ≥9 mots (5/100)	91.00%					
	Nn			99%	33-41	3	NeoJ: six, RF:2,	75%

Adm: Admission, HL Ago fr Adm: How long ago from admission, M cared C: Mother cared child, Rel cared C: Relative cared child, GA (Ws): Gestational age (Ws), NI: Neonatal infection, NICU adm Bef FS: NICU Admission before fecal sampling, Reas for NICU ADM: Reasons for NICU admission, Del M - VAG Del: Delivery method- Vaginal delivery, NeoJ: Neonatal Jaundice, PTB: Premature birth, RF: Respiratory failure, BLDS: Bloody stool

Table 6. Characteristics associated with CPE in mothers and neonates (Cont.2)

Occup. (1)	Study Participants (2)	M: Chronic diseases (cases) (17)	M: Sx Op Bef P in CPE study (cases) (18)	Characteristics of M: Dur of T from HV ADM to SC (hours) (19)	Characteristics of M with CPE colonization: Dur of T from HV ADM to SC (hours), n=21 (20)	Characteristics of N with CPE colonization: Dur of T from Del to SC (hours), n = 13, (21)
HCWs (n=8)	Mother	No	1	Mean = 20.37 hrs, Std. Deviation = 45.77 hrs, Min = 0 hrs, Max = 549 hrs (22.87 days), 86.60% (from 0 to 28.00 hrs)	Mean =33.76 hrs, Std. Deviation = 46.35 hrs, Min = 0 hrs, Max =159.00 hrs (6.63 days), 76.20% (from 0 to 28.00 hrs)	Mean =40.07 hrs, Std. Deviation = 4.462 hrs, Min = 31 hrs, Max =47.00 hrs (1.95 days), 100% < 48 hrs
State Employee (n=173)	Mother	18	10			
Businessperson (n= 51)	Mother	2	6			
Self emloyee (n= 27)	Mother	1	3			
Stay-at-home spouse (n = 100)	Mother	5	7			

OCCUP: Occupation, M: Mother, M: Sx Op Bef P in CPE study: Mother: surgery operation before participating in CPE study, M: Dur of T from HV ADM to SC (hour)s: Mother: duration of time from HV Hospital admission to sampling collection, Characteristics of M: Dur of T from HVADM to SC (hours): Characteristics of Mother: Duration of time from HV admission to sampling collection, Characteristics of M with CPE colonization: Dur of T from HV ADM to SC (hours): Characteristics of Mothe with CPE colonizationr: Duration of time from HV admission to sampling collection, Characteristics of N with CPE colonization: Dur of T from Del to SC (hours): Characteristics of Neonatal with CPE colonizationr: Duration of time from HV admission to sampling collection (hours)

Table 7. Characteristics associated between labor induction in mothers and the length of time from labor induction to sample collection over 36 hours

Characteristics	Freq.	Percent
1. Total of antibiotic doses		
no dose	1	14.3
≤2 doses	5	71.4
≥3 doses	1	14.3
2. Prevalence of CPE		
Non-CPE	4	57.1
CPE	3	42.9
3. Length of time from labor induction to sample collection		
>36-48 hrs	2	28.6
>48 hrs	5	71.4
4. Association between 2 and 3		
Length of time from labor induction to sample collection	Freq of CPE	
>36-48 hrs	1	100
>48 hrs	2	100
5. Association between 1 and 2		
Antibiotic use	Freq of CPE	
Yes (≤2 or ≥3 doses)	3	100
6. Length of time of Hospital stay Before Sample collection		
>48 hrs	7	100

compared with that of from 1-5 times, with P = 0.041, OR = 14.487, 95% CI: 1.115-188.237, as shown in the Characteristic 9 of Table 5.

Source of CPE colonization in mothers

Based on the results of prevalence and risk factors associated with CRE colonization, displayed in Tables 1, 2, and 3. Table 6 lists participants' professions and shows that the prevalence of CPE was 0% (n = 8), 6.40% (n = 173), 5.90% (n = 51), 7.40% (n = 27), 5.00% (n = 100) in the group of HCWs, State employees, Businesspersons, Self-employed persons, and Stay-at-home spouses, respectively. The result of Table 6 was surprising, as CPE did not appear in HCWs. However, we detected CPE in state employees and Housewives, who had the potential to contact hospital environments and healthcare, fewer than the HCWs, a key characteristic of all CRE-colonized mothers, especially CR. *E. coli* colonized mothers did not acquire any hospital infections, any chronic disease, or any surgical operation before participating in this study (Table 6).

Moreover, one impressive character was that the duration from hospital admission at the study site to the fecal sample collection in mothers with CPE colonization was 76.20% (from 0-28.00 hrs) (Min: 0 hours, and Max = 159.00 hrs (equal to 6.63 days).

In neonatals

Table 6 showed that the duration of time from delivery to sample collection in the neonatal colonized with CPE was 31.00-47.00 hours. The result in Table 6 confirmed one case of non-hospital-acquired and non-CPE caused infection determined in one neonate.

Duration of time from labor induction to fecal sample collection

Table 6 showed that the duration of time from delivery to sample collection in the neonatal colonized with CPE was 31.00-47.00 hours, and only one case of non-hospital-acquired and a non-CPE caused infection was a neonatal infection found in one neonate.

Association between the duration of time from labor induction and fecal sample collection over 36 hours

Table 2 showed that labor induction and the duration of time from labor induction to sample collection (more than 36 hours) were risk factors for CPE colonization. Specifically, the group of pregnant women (n = 7), who spent time from labor induction to sample collection (over 36 hours, had a CPE colonization prevalence at 42.90% (3/7). Three of six pregnant women who consumed antibiotics had CPE colonization, with 50% (3/6).

DISCUSSION

The results described in Table 1 suggested how to determine the origin of KPC in neonates and distinguish *K. pneumoniae*-producing carbapenemase in mothers and children. It was whether Carbapenemase-producing *E. coli* found in mothers was the same source in neonates. Could the origin of *K. pneumoniae* and *E. coli* in

mothers and neonates be from the community, the hospital environment, healthcare workers, or other sources that did not belong to the hospital?

Source of CPE colonization in mothers

As shown above, the duration from hospital admission at the study site to the fecal sample collection in mothers with CPE colonization was 76.20% (from 0-28.00 hrs), and characteristics of mothers with CPE colonization: The duration of time from admission to sample collection (hours) in pregnant mother acquired CPE colonization, was 76.20% (from 0-28.00 hrs), Table 6, (Cont.2). Thus, was this length of time enough for the CPE to exist in the gastrointestinal tract of the mothers? A case-control study reported that duration of hospital stay over 28 days (OR 23.6, 95% CI 4.9-113.3) in the last 12 months was a risk factor for acquiring *K. pneumoniae* producing carbapenemase, detected with rectal swabs.[18] Moreover, another case-control study reported that hospital stays exceeding twenty days were a risk factor for acquiring carbapenemase-producing *Enterobacteriaceae*, as determined similarly with rectal swabs (AOR: 4.9, 95% CI: 1.4-15.5; $P < 0.001$).[19] The duration of CPE detection in the two studies above showed that CPE found in mothers in our research could appear before admission to Hung Vuong Hospital or be due to cross-contamination happening at the study site.

We recognized that, for example, the hospital admission before study participation about one year, the number of days for hospital stay in the last admission, chronic diseases, past surgical operation before study participation, professional, and duration of performance of professional (years) weren't risk factors of CPE colonization in our study. Hence, the CPE detected in mothers in our study could belong to the community where they have lived. For example, multidrug-resistant organisms can spread to members of the same household. Researchers also reported that ESBL carriers can transmit genetically related ESBL-producing bacteria to their family members, with a prevalence from 17%-32%.[20-25]

Source of CPE colonization in neonatals

In Table 6, fecal sample collection in the neonatal colonized with CPE was from 31.00 to 47.00 hours after delivery. This result showed that CPE colonization in neonates could originate from potential contamination between the hospital environment, healthcare practices, and the neonates.

CPE transmission between mothers and neonates

The result of evidence of CPE transmission, shown in Figure 2, suggested that CPE transmission between this mother and her baby might be associated with the vaginal canal, which supplied an environment suitable for developing the colonization of pathogenic bacteria, which might move ascendingly into the uterus of mothers after the event of the premature rupture of the amniotic membrane. The result of this event was the contamination of amniotic fluid. Consequently, if the neonate inhaled or swallowed the infected amniotic fluid, pathogenic bacteria would colonize in their gut. Then, they would cause intrapartum sepsis, as studies reported *K. pneumoniae* found in the neonate's gut is the principal pathogen of intrapartum sepsis.[26-28] Although the CPE transmission prevalence was very low in our study, it confirmed that the CPE transmission issue should be a concern in the future due to the pressure of antibiotic resistance caused by CPE as a problem in developing countries.[29] For example, a study in Algeria conducted on all mothers and their newborns in two maternity units, including Bejaia (Maternity A) and northern Algeria (Maternity B) in Tizi Ouzou, was prospective and randomly recruited from 357 mothers and 365 newborns from January 1 to April 30, 2017. This study reported a mother-newborn pair carrying asymptomatic OXA-48-producing *E. coli* and *K. pneumoniae* isolates.[30] The researchers in Algeria also suggested that the CPE of mothers could contaminate their neonates, or neonates' CPE could be from other sources.[30]

Risk factors of CPE colonization in mothers

The results of the univariate binary logistic regression associated with Risk factors of CPE colonization in mothers suggested the question was whether the time from labor induction to fecal sample collection with the rectal swab over 48 hours influenced CPE contamination in mothers. This question is essential and valuable

in controlling and preventing CPE transmission or contamination in mothers because it will be a new direction in determining the factors related to CPE contamination or transmission for mothers spending hospital stays for delivery because in our study, one positive factor influencing CPE colonization was the mothers' age from 19-23 years old, which reduced the CPE colonization compared with the age of mothers, less than or equal to 18 years old, with P = 0.033, OR = 0.067, 95% CI: 0.006-0.802 (Table 2).

One significant detail we analyzed above, the associations between the duration of time from labor induction to fecal sample collection over 36 hours was the risk factor of CPE colonization at 42.90% (n = 7), and one impressing finding is that mothers, who consumed antibiotics (less than two doses or more than three doses), and stayed in the hospital for over 48 hours before fecal samples were collected, they would acquire CPE colonization (Table 7).

The result of the multivariate binary logistic regression of risk factors of CPE colonization in mothers noted that the mothers had a history of antibiotic use before study participation, with the total number of antibiotic doses (1-2 doses) in the period of hospital stay being the principal risk factor of CPE colonization compared with mothers without antibiotic use.[31] This result showed that maternal CPE colonization could happen before hospital admission. CPE colonization could appear in the community before pregnant women's admission to Hung Vuong Hospital. The origin of maternal CPE colonization could be the antibiotic overuse in the community, where over-the-counter antibiotics can be bought at pharmacies without a doctor's prescription, as some research reported in Vietnam.[32]

The overuse of antibiotics in livestock has been a significant challenge in controlling multidrug-resistant organisms in Vietnam, as evidenced by the presence of antibiotic residues in food, such as pork and chicken meat.[33-35]

Antibiotic residue could be one of the causes of bacterial antibiotic resistance in the human gastrointestinal tract, leading to the development of multi-resistant organisms.[36] Hence, maternal CPE colonization could be present in the maternal gastrointestinal tract before the time of pregnant women's hospital admission.

Risk factors of CPE colonization in neonates

The results of the univariate binary logistic regression of risk factors of CPE colonization in neonates showed that admission to the Neonatal Intensive Care Unit before the fecal sample collection performed on neonates was a risk factor for CPE colonization, as shown above (Table 4). The Neonatal Intensive Care Unit was a potential source of CPE contamination. At a Portuguese NICU, a prospective, observational, longitudinal, and cohort study reported that the prevalence of CPE isolates detected in 173 admitted neonates was 5.8%, including the most frequent carbapenemase-producing *Klebsiella pneumoniae* isolates.[37] Next, another study in a neonatal intensive care unit in Morocco reported that during hospitalization, 12.5% (26/207) of them acquired CPE that exclusively expressed the bla_{OXA-48} gene.[38] The study results, as described above, including our findings, showed neonates might acquire CPE colonization through the cross-infection route in which CPE-colonization source can present in HCWs, neonates' relatives, or in hospital environments, and not in mothers because the from-mother-to-neonate transmission prevalence of 0.28% (1/359) (*E. coli*) was very low. It meant that NICU-admitted neonates, before the fecal sample collection, were at risk of being colonized by CPE more than other neonates without a NICU admission history. Hence, the NICU admission was a potential risk factor for CPE colonization in our study.

Impressively, our study result also showed that CPE colonization detected in neonates had a history of NICU admission for over 17 hours, while A retrospective case-control study conducted by matching pairs based on the date of birth and gestational age to determine the ESBL-colonized infants at the NICU of Soroka University Medical Center, Israel from 2013-2014 proved the mean time from admission to ESBL-producing-bacteria colonization was 15 days.[39] Our study results suggested a potential CPE contamination occurred between HCW, or the hospital environment, and neonates in

the NICU. The former studies reported that the areas used for CPE patients to stay in the hospital were significantly contaminated.[37,38,40] Hence, we should collect neonates' fecal samples at the beginning of NICU admission to detect early CPE colonization in neonates admitted to NICU and have a plan to prevent and control CPE colonization in neonates during hospital stay. Significantly, as reported in our results (Table 4), vaginal examination was associated with CPE colonization in neonates. Our study showed that the number of vaginal examinations from 6-9 was a risk factor for CPE colonization, as shown in Table 4. The risk of bacterial colonization in the first vaginal examination in pregnant women was 0.34 times lower than that in more than one vaginal examination in women (OR 0.34; 95% CI 0.241, 0.481).[41]

This result implied that good medical practices have become a crucial standard in preventing CPE colonization/transmission from HCWs or the hospital environment to mothers in healthcare settings. For example, some studies reported that the transmission from HCWs was more significant in the areas where the nosocomial outbreak appeared.[42] Two prospective studies determined the HCW-to-patient transmission route of the unique MRSA clones. Another study demonstrated that the HCWs' hands were the source of *S. aureus* transmission from patient to patient, reported in three US academic medical centers.[43,44]

Next, A significant and impressive result is that the benefit of breastfeeding in our study was to reduce CPE colonization in neonates. A few studies showed this benefit. For example, trials reported that breastfeeding is beneficial for children because breastfed children had a lower frequency of antibiotic use than unbreastfed children. There was a strong relationship between the duration of breastfeeding and the frequency of antibiotic use in children.[45,46] Moreover, breastfeeding was a beneficial factor in preventing acquisition of ESBL-producing *Enterobacteriaceae*, with an adjusted hazard ratio of 0.29 (95% CI, 0.11, 0.80).[47]

Another question is how the risk factors influenced each other. It is impressive that we analyzed the result of multivariate binary logistic regression of risk factors related to CPE colonization in neonates (Table 5). We found that the number of vaginal examinations from 6-10 times was the principal risk of CPE colonization. Another study reported that the risk of bacterial colonization from the first vaginal examination in pregnant women was 0.34 times lower than that from more than one vaginal examination (OR 0.34; 95% CI 0.241, 0.481).[43] This result suggested that limiting the number of vaginal examinations is the best solution to reduce CPE colonization in neonates.

CONCLUSION

Antibiotic consumption before fecal sample collection and the duration from labor induction to sample collection were risk factors for CPE colonization in pregnant women. At the same time, the beneficial characteristics for reducing CPE colonization in neonates included the vaginal delivery, mother caring for neonates, gestational age over 37 weeks, less than two relatives caring for neonates, two hours for skin-to-skin contact between mothers and breastfeeding, Especially, the interaction between two characteristics, including vaginal delivery, mother caring for neonates reduced the CPE colonization in neonates.

The findings of mother-to-neonate CPE transmission were significantly low. However, the trend of CPE transmission in the group of mothers and neonates, and CPE-colonized mothers targeting interventions with screening CPE colonization at the beginning of hospital admission is the best solution to reduce this cross-transmission in endemic settings, and hand hygiene compliance and aseptic medical practices are crucial standards to prevent and control CPE in the healthcare sector.

Ultimately, the use of over-the-counter antibiotics in the community and the misuse of antibiotics in livestock farming need to be closely monitored by government regulations. The Government opens communication channels to guide and educate the community on when to use antibiotics and how to use them when prescribed

by a doctor. It shouldn't use antibiotics as a means to promote livestock growth. These procedures will reduce bacterial antibiotic resistance, including multidrug-resistant organisms, CPE.

Limitation of study

As described, we did not collect the fecal samples of HCWs and relatives of neonates and environmental samples to determine the transmission between neonatal CPE colonization and HCWs, or neonates' relatives or environment.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

AUTHORS' CONTRIBUTION

NVK conceptualized the study. PTH contributed to preparing the questionnaires. TTTH, NTKN and HNP collected data. CTT, NVMH, JIC and TTBC performed the experiments. NMN and NVK analyzed the data. NVK wrote the manuscript. SB and PTH reviewed and revised the manuscript. All authors read and approved the final manuscript for publication.

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DATA AVAILABILITY

All datasets generated or analyzed during this study are included in the manuscript.

ETHICS STATEMENT

This study was approved by the Board of Hospital Directors and the Ethics Committee of Hung Vuong Hospital.

INFORMED CONSENT

Written informed consent was obtained from the participants before enrolling in the study.

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